The treatment of early syphilis should be fairly routine, complications alone suggesting variations or modifications. Drug intolerance and the condition of the patient, of course, require medical judgment. Regular and continuous treatment are most important in any plan, and error toward overtreatment, rather than too early discontinuance, is preferable.

Epidemiologically, the case of early syphilis is, of course, most important. With an estimated syphilitic population of around 500,000 new active cases in the United States on any given date, and with 5 to 10 per cent of any urban group infected, we should certainly be concerned with the preventive aspect of this truly major communicable disease. All investigations would seem to indicate that the incidence does not vary from time to time, although urban groups seem to have a higher incidence than rural communities. Propaganda stimulating general use of prophylaxis has aided the European countries (notably England and Sweden) in reducing the number of new cases. Any good treatment plan should aim not only to cure the patient, but to shorten the period of infectiousness and thus limit the spread. The disease has become one of the major causes of death, and must be seriously combated by the public health authorities, aided by the physicians. Due to an unfortunate association of terms, syphilis is regarded entirely as a venereal disease, with the moral connotation attached to the word "venereal." With 7 per cent of our cases extragenital, and with innocently acquired marital infections and congenital cases making up nearly one-half of the total, it is time our people were educated to separate this disease from vice, prostitution and immorality. The euphemism of "social disease" should be discarded and cases reported by name and address, just as any other communicable disease. Not unlike tuberculosis, it should be considered by the public in much the same light. Only then can epidemiological studies be carried through to completion and the spread of the disease adequately combated. Adequate follow-up service is essential, not only to control the spread but to complete the treatment. Education of the patient in the necessity of continuous therapy is important. Health Departments should provide adequate treatment of indigents, should acquire accurate morbidity statistics through laws requiring the reporting by name and address, and should do more thorough epidemiological work, including quarantine of acute cases when necessary, ignoring the moral and police responsibilities often associated with the disease.

During the past generation we have seen the possibilities of publicity campaigns in public health efforts toward combating and controlling contagious diseases. Look at what has been accomplished in eradication of smallpox, control of diphtheria, and practical elimination of typhoid fever. Recently the efforts in poliomyelitis control were handsomely rewarded by public interest and coöperation. Cancer has reached the public mind as never before, entirely because of frank discussions and educational efforts with the people.

Such papers as Doctor Chambers has written will do much in bringing to the general medical practitioner a new stimulus to diagnose early and institute at once adequate treatment, and it is in the hands of the general profession that the future attack lies, as most patients consult the specialist only later.

❦

Norman N. Epstein, M. D. (450 Sutter Street, San Francisco).—A clear understanding of the principles involved in the diagnosis and treatment of syphilis in the early period is essential if the patient is to be given the greatest opportunity of cure and if the spread of this disease is to be checked. Early syphilis, that is, the time extending from the inoculation with the Treponema pallidum through the disappearance of secondary symptoms, is highly contagious, and at the same time most amenable to treatment. Therefore the earlier the disease is detected and treated, the better for the individual and the general community.

A recent comprehensive survey by the Coöperative Clinics, under the direction of the United States Public Health, shows that with the best-known treatment 83 to 86 per cent of patients in the seronegative primary stage 64 to 70 per cent of patients in the seropositive primary stage, and 61 to 82 per cent of patients in the secondary stage can be clinically cured. This period then is, as Doctor Chambers says, the golden opportunity for treating syphilis.

It should be emphasized that the continuous method of treatment is the most satisfactory from many standpoints. In the early phase of syphilis, particularly the primary stage, the patient has developed little or no resistance to the Treponema pallidum, and the use of highly treponemicidal drugs such as the arsphenamins does not permit the resistance mechanism of the body to act. Therefore, cure of the disease in this phase must be accomplished by drugs alone. If insufficient therapy is administered, the patient is very apt to develop the various forms of recurrence, especially neurorecurrence and mucous membrane recurrence. This fact must be energetically impressed upon the patient in order that he do not discontinue treatment before his year and one-half, or two years have elapsed.

We agree with Doctor Chambers in his plan of treatment, except in the matter of dosage. We do not use more than 0.6 grams of neoarsphenamin, and our initial dose is 0.45 grams. Where a severe Herxheimer reaction may be anticipated, one injection of bismuth precedes the neoarsphenamin by twenty-four hours. We have found that this dosage is adequate and less likely to produce toxic reactions, which seriously interfere with carrying out the general plan of treatment.

Doctor Chambers has presented an excellent review of the treatment of early syphilis, and his paper is of importance to all practitioners.

ACUTE PERFORATION OF GALL-BLADDER WITH GENERALIZED CHOLEPERITONEUM*

By E. Eric Larson, M.D.
Los Angeles

Discussion *by Loren L. Chandler, M. D., San Francisco; Harlan Shoemaker, M. D., Los Angeles; and Stanley H. Mentzer, M. D., San Francisco.*

ACUTE perforation of the biliary system occurs infrequently, is rarely diagnosed, and carries a very high mortality. Several authors have reported series of acute gall-bladder rupture, but undoubtedly many widely scattered occurrences of this condition remain unrecorded. Acute gall-bladder rupture is much more frequent than acute rupture of the biliary ducts. Recently Wolfson and Levine reported three instances of spontaneous rupture through an area of infection and weakness of the common bile duct following cholecystectomy and common duct exploration. That the acute, tense, infected, and obstructed gall-bladder ruptures with such infrequency, is due to a rich blood and lymph vascular system and the tense fibromuscular wall of the organ. The reduced virulence of bacteria by the action of the bile and the distensibility of the cystic duct must also be important factors.

TYPES OF ACUTE PERFORATIONS

The several large series reported by various authors include all types of acute perforations. These should be divided into three distinct groups. The diseased gall-bladder of long standing which has built about itself adhesions to surrounding viscera, such as omentum and coils of intestines,

* Read before the General Surgery Section of the California Medical Association, at the sixty-fourth annual session, Yosemite National Park, May 13-16, 1935.

CHART 1.—*Acute Perforation of Gall-Bladder with Generalized Choleperitoneum*

	Name	Sex	Age	Occupation	Result	Autopsy
1.	A. H.	F	60	Maid	Recovery	.....
2.	O. I.	M	52	Laborer	Death	Done
3.	M. M.	M	47	Laborer	Death	Done
4.	L. A.	F	56	Housewife	Recovery	.....
5.	E. A.	M	21	Laborer	Death	Not done
6.	T. O.	M	75	Laborer	Death	Done
7.	S. M.	M	56	Laborer	Recovery	.....
8.	K. K.	M	62	Laborer	Death	Done
9.	J. F.	M	60	Laborer	Recovery	.....
10.	I. R.	F	49	Housewife	Recovery	.....
11.	R. K.	M	58	Laborer	Recovery	.....
12.	S. M.	F	59	Nun	Death	Not done
Recovery: 50 per cent.			Death: 50 per cent.		Autopsy: 66+ per cent.	

becomes, when it ruptures, a localized disease with characteristic manifestations. Recognition of an infected mass in the upper right quadrant, associated with a classical history of an acute gall-bladder attack with an antecedent gall-bladder history, should make one suspicious of the nature of the existing pathology. The patient can be adequately prepared for surgery and the plan of attack well outlined.

The second group includes those patients in whom the acutely diseased gall-bladder ruptures and drains by fistulous tracts, either into a neighboring viscus or through the abdominal wall. Abel reports a case with the external opening below Poupart's ligament, and Bye reports a fistulous tract extending to the right flank. These patients can also be critically studied and properly prepared for surgical attack. The third and most distressing type of acute perforation of the biliary system is that in which the peritoneal cavity is flooded with visceral contents, producing a choleperitoneum which gives rise to peritonitis and toxemia. This group taxes the diagnostic acumen and display of surgical judgment, since immediate interference is mandatory. The mortality in this group ranges between 50 per cent and 100 per cent. This report will deal with twelve patients who belong to this last group. Nine of these histories are from the records of 706 gall-bladder operations, with seventy-six deaths at the Los Angeles General Hospital. Two are from the records of 353 gall-bladder operations at St. Vincent's Hospital, and one was under my care surgically at the Woodland Clinic, September, 1926. One of the patients from St. Vincent's Hospital was surgically treated by Dr. W. R. Molony, Sr., and the author, recently. (See Chart 1.)

CAUSE OF ACUTE RUPTURE PRODUCING CHOLEPERITONEUM

Acute rupture of the gall-bladder producing choleperitoneum may occur from several causes,

CHART 2.—*Series of Gall-Bladder Ruptures Reported by Twelve Authors*

Year	Author	Number of Cases	Remarks	Mortality
1890	Courvoisier	34	(Following trauma)	65 per cent
1903	Erdman	34	(During typhoid fever)	97 per cent
1905	Ricketts	273	64 operated on—21 died 154 died—160 not operated	56 per cent
1912	McWilliams	108	All surgically treated	48 per cent
1918	Buchanan	17	12 treated surgically	50 per cent
1925	Georg	348	.....	42 per cent
1926	Gosset	111	33 per cent calculous gall-bladders	52 per cent
1926	Fifield	27	96 per cent calculous gall-bladders 22 drained	44 per cent
1927	Alexander	20	60 per cent calculous gall-bladders 14 drained only	35 per cent
1928	Mitchell	16	69 per cent calculous gall-bladders 5 drained only	43 per cent
1934	Eliason McLaughlin	9	89 per cent calculous gall-bladders 8 drained only	11 per cent
1935	Larson	12	85 per cent calculous gall-bladders One anaerobic infection	50 per cent

CHART 3.—*History Notations*

Previous Indigestion	Character	Pain Severity	Radiation	Shock	Vomiting
1. Several years	Sudden	Sharp	None r. u. q.	Moderate	Constant
2. Several years Acute gall-bladder three weeks	Constant	Extreme	.....	Moderate	Intermittent
3. Several years	Sudden	Very severe	Right lumbar	Extreme	Once
4. Several years	Sudden	Very severe	Right shoulder Epigastrium	Moderate	Intermittent
5. None	Severe	Marked	None	Extreme	.....
6. Much	Constant	Marked	Epigastrium	Moderate	.....
7. Years	Constant Sharp	Moderate	.....	Some	Five or six times
8. Years	Constant five days	Marked	.....	Present	None
9. Sudden, 1:30 a. m.	Sudden r. l. q.	Extreme	Right shoulder Right neck	Marked	Present
10. Sudden, 7 a. m.	Sudden	Agonizing	.....	Moderate	Present
11. Years	Sudden Cramping	Moderate	Costal: Epigastrium Right shoulder	Moderate	Present
12. Vague, for years	Sudden 7 a. m.	Marked	.....	Marked	.....

the most usual being cholecystitis associated with cholelithiasis. Violence, such as blows or piercing thrusts with sharp instruments, most notably stab-wounds, may result in choleperitoneum. Spontaneous rupture of the viscus may occur because of weakness of the gall-bladder wall, secondary to thrombosis of the vascular system or ulceration resulting from pressure of gall-stones. Ulceration due to infection, most notably typhoid or streptococcus, may lead to spontaneous rupture of the viscus. The perforation is usually at or near the fundus, and is found most commonly in women, they having a greater susceptibility to gall-bladder disease. It may happen at any decade of life, Power and Johnston reporting such an occurrence in a child two years of age due to empyema of the gall-bladder associated with ascaris lumbricoides. Most instances fall in the latter decades of life.

Chart 2 represents series reported by twelve authors including the number of patients, salient remarks and mortality. All these series contain instances of gall-bladder ruptures of the three main types. The writer's series is included in the third group, representing rupture with generalized biliary peritonitis.

DIAGNOSIS

Acute gall-bladder rupture with choleperitoneum is comparable to the perforation of any viscus, and since it is spontaneous, the diagnosis is most

CHART 4.—*Examination and Working Diagnosis*

Abdominal Rigidity	Distention	Peristalsis	White Blood Count	Preoperative Diagnosis
1. Epigastrium and r. u. q.	Slight	.....	16,800	General peritonitis from appendix or ruptured peptic ulcer
2. Present	Mild	.....	.....	Intestinal obstruction
3. Present	Present	Not stated	16,500 polys 80 per cent 18 hours later 21,500 and 86 per cent	Perforated peptic ulcer? Ruptured appendix?
4. Present	Mild	Not stated	17,500 polys 86 per cent	Ruptured gall-bladder
5. Present	Present	.....	Not made	Abdominal stab wound
6. Present	Present	None	Not made	Abdominal malignancy Possible cancer head of pancreas Possible obstruction of common duct Addison's disease (jaundiced)
7. None	None	Not stated	6,200	Gall-stones Acute cholecystitis
8. Present	Present	None	.....	None made
9. Present r. l. q.	Present	Present	15,400 polys 92 per cent	Perforated ulcer or ruptured appendix
10. Extreme	.....	.....	22,000 polys 96 per cent	Perforated peptic ulcer
11. Marked	Marked	.....	17,000 polys 93 per cent	Ileus, cause unknown
12. Marked	None	.....	13,200 polys 78 per cent	Perforated peptic ulcer

CHART 5.—*Pathology*

Perforation	Size	Position	Gall-stones
1. Edges necrotic	1½ centimeters	Fundus	Present
2. Gall-bladder gangrenous	.....	Fundus	Present
3. Ragged	4 millimeters	Medial wall	Present: smear B. coli
4. Ragged	1½ centimeters	Medial wall	Present in cystic duct
5. Knife stab	.....	Fundus	None
6. Tear	Gall-bladder almost destroyed	.....	Stones in common duct; duct 2 centimeters in diameter: cancer head of pancreas
7. Ragged	Not stated	Medial wall	Present
8. Ragged ulceration	1½ centimeters	Fundus	Present in cystic duct: common duct stones
9. Rupture	Not stated	Not stated	Present
10. Rupture	⅔ centimeter	Fundus, lateral side	Present: outside of gall-bladder
11. Ragged	1 centimeter	Fundus	Present
12. Ragged	1 centimeter	Cystic duct	None

confusing. The patient presents the picture of a sudden severe intra-abdominal catastrophe with diffused abdominal symptoms. (See Chart 3.) In the very early stages of the disease a perforated peptic ulcer is usually suspected. The next most frequent diagnosis is a ruptured, high-lying appendix with general peritonitis. Cystic duct obstruction with empyema of the gall-bladder is commonly suspected. Later the distention, pain, and vomiting may lead to the diagnosis of an intestinal obstruction with ileus. On the other hand, acute pulmonary infections or coronary thrombosis may mislead one to the erroneous diagnosis of an unusual gall-bladder syndrome. However, if the history indicates a long standing biliary disease antedating acute cholecystic disease, becoming complicated by a sudden severe pain in the upper abdomen and followed by generalized abdominal signs of peritonitis, a perforated gall-bladder with choleperitoneum should be suspected. A mild rise in temperature, with an increased white blood count, aids in forming a correct diagnosis. Rigidity, rebound tenderness, and distention are usually present. (See Chart 4.) The patient gives evidence of a generalized peritonitis. The type of bacteria, if present and released with the bile, may alter the degree of severity and toxemia of the peritoneal irritation.

TOXIC EFFECTS OF FREE BILE IN THE ABDOMINAL CAVITY

It is of interest to review some of the experiments that have been done relating to the toxic effects of the bile free within the abdominal cavity.

Frankel and Krause, in 1899, injected sterile bile into the peritoneal cavity of guinea-pigs and dogs without any untoward effects. Rents in the gall-bladder produced experimentally were proven healed in two to six weeks later, at necropsy. There was no obstruction to the free flow of bile in the common duct.

Wangensteen, in 1926, ligated the common duct and cut a hole in the gall-bladder in six dogs and later in rabbits, with death within twenty-four hours. He concluded that death was due to the toxicity of the bile.

Horrall, in 1929, confirmed the findings of Wangensteen. He concluded that continuous drainage of bile into the peritoneal cavity is rapidly fatal because of the toxicity of the bile salts.

Since bile peritonitis was considered a toxemia, Rewbridge, in 1931, made some experiments and concluded that bile free in the abdomen of dogs produced in every instance an invasion by B. Welchii. This was presumably the result of changes in the intestinal wall, due to increased permeability caused by the action of the bile salts. He also found that blood examinations for bile salts and bilirubin are of no value in determining the amount of drainage of bile into the peritoneal cavity. He concluded that the toxemia is due to a peritonitis by B. Welchii, and not to bile salts.

Since all herbivorous animals and only a few humans have the anaerobic organisms within the gastro-intestinal tract, one would expect complications of anaerobic infections in humans to be most unusual. In this series, anaerobic infection was encountered only once. The patient, number eleven (Chart 1), developed gas-gangrene throughout the entire length of the wound and adjacent tissues. The condition was treated by wide exposure and constant irrigations with an oxidizing solution. A good recovery resulted except for a marked incisional hernia.

TREATMENT

The treatment embraces two very important features: The early suspicion or definite recognition of the disease, and the immediate surgical interference. Preoperative and postoperative care of these patients demand unfailing attention. In contrast to perforated peptic ulcer, we have in choleperitoneum an immediate bacterial invasion of the peritoneum, while the contents following perforation of a peptic ulcer are usually rendered sterile for many hours by the action of the gastric

CHART 6.—*Operations*

Anesthesia	Condition	Procedure
1. Ether	Fair	Cholecystectomy: local and pelvic drainage
2. Ether	Poor	Cholecystostomy: three rubber drains Kidney foramen and between
3. Ether	Poor	Cholecystostomy: local drainage
4. Ether	Fair	Cholecystostomy: local drainage
5. Ether	Poor	Repair of laceration
6. No operation	.....	.....
7. Ether	Fair	Cholecystectomy: local drainage
8. No operation	.....	.....
9. Local, 150 milligrams	Poor	Cholecystostomy: local drainage
10. Spinal, 200 milligrams	Poor	Cholecystectomy: local drainage Two tubes in pelvis
11. Ether	Poor	Cholecystostomy: local drainage
12. Ether	Poor	Cholecystostomy: local drainage

hydrochloric acid. With the abdomen open and free bile widely scattered, the surgeon must exercise good operative judgment in his procedure. The perforation is easily found. However, Nogués and others have reported choleperitoneum without finding an opening, assuming the bile to be a transudate from the biliary system. The perforation (Chart 5), as stated previously, is usually at or near the fundus, although in one instance it was localized in the cystic duct. Gall-stones usually present in these patients may be free in the abdominal cavity, and must be looked for and removed, since they are foreign bodies and may lead to complications. Cultures should always be made, since anaerobic organisms may be present, as in one of this series. The question of good toilet and simple cholecystostomy with adequate drainage, or cholecystectomy, with new areas for absorption opened up, must be determined. Cholecystostomy, with as little trauma as possible, and perhaps an ileostomy in the delayed and extremely toxic patients, may be safest. The age of the patient, his general condition, age of the perforation, and laboratory data, as well as the skill of the surgeon, must be the deciding factors. A cholecystectomy

CHART 7.—*Acute Perforations of Gall-Bladder*

1. Perforation	Process remaining localized—by adhesions, omentum and coils of intestines.
2. Perforation	Fistulous tract communicating between gall-bladder and gastro-intestinal tract or through the abdominal wall.
3. Perforation	Gall-bladder contents scattered through the abdominal cavity. (Choleperitoneum.)

CHART 8.—*Summary*

1. Ruptured gall-bladders fall into three distinct types in regard to diagnosis, surgical pathology, and treatment. The first type concerns those that remain localized. The second establishes fistulous tracts for drainage and the third floods the peritoneal cavity with the contents of the biliary tract. This paper reports twelve instances of the third group. Ten were surgically treated. The mortality was 50 per cent. Two not surgically treated died.
2. The diagnosis is difficult. It was made preoperatively in only one instance. Eternal vigilance must be exrcised in all delayed treatment in acute gall-bladder disease for the recognition of perforation, because early surgical interference is mandatory.
3. Culture of peritoneal fluid for type of bacteria present is urged. Anaerobic organisms may be present and operative procedure, therefore, altered. It is generally considered that cholecystostomy and adequate drainage is safer than cholecystectomy. In this series cholecystostomy was done seven times, cholecystectomy three times.
4. One patient's recovery was complicated with anaerobic infection of abdominal wall with recovery.

can perhaps be more safely done later. We know so little regarding liver function and liver shock that conservative measures seem most important especially in an obese patient or one having suspected myocarditis. Even though the bile may be sterile, the trauma connected with cholecystectomy may be severe enough to cost the life of a patient. In this series cholecystectomy was done three times, cholecystostomy seven times.

The postoperative care of these patients embodies the same principles as used in any case of peritonitis. Vigilance in the supply of sufficient fluids, such as glucose and saline by intravenous or subcutaneous routes, is important. Adequate morphin for pain and preservation of intestinal muscular tone is desired. Drainage of such secondary intra-abdominal collections as often occur in the pelvis may be necessary.

The presence and character of any bacteria in the bile may, of course, modify any of these procedures.

SUMMARY

1. A series of twelve patients with generalized biliary peritonitis following ruptured gall-bladder is reported. Ten were operated upon and two treated medically. The mortality was 50 per cent.

2. Rupture of the viscus must be constantly kept in mind in all instances of acute gall-bladder disease. Early recognition and prompt surgical measures are mandatory.

3. The ruptured gall-bladder that remains localized or forms fistulous tracts for drainage is more readily diagnosed and more safely cared for surgically.

4. Cholecystostomy with efficient drainage is safer than cholecystectomy.

5. Ileostomy should occasionally be utilized. Culture of the peritoneal exudate is urged. Sterile bile is "devoutly to be wished."

1930 Wilshire Boulevard.

DISCUSSION

LOREN L. CHANDLER, M. D. (Stanford Hospital, San Francisco).—Acute perforations of the gall-bladder are not so infrequent as many people presume, and

if not recognized and treated promptly, they almost always result fatally. Doctor Larson has presented this subject exceptionally well. His plea for adequate bacteriological studies at the time of operation cannot be supported too strongly, for the character of the bacterial infection undoubtedly influences the course of most of these cases. Our experience at Stanford Clinic is not unlike Doctor Larson's; but, in reviewing these cases, I note one complaint common to all—the sudden onset of sharp, severe pain in the right lower quadrant. It is reasonable to suppose that the exudate from the perforated gall-bladder would be kept away from the peritoneum of the anterior abdominal wall by the liver, transverse colon and omentum, but it would contact the peritoneum in the middle or lower abdominal quadrant, manifesting itself by severe pain in this area. All of our cases had cholelithiasis, obviously of long standing. Two of the gall-bladders were carcinomatous.

An accurate diagnosis of perforated gall-bladder is difficult, but I wish to call attention to the value of the Schilling differential blood count. This is done by differentiating the young polymorphonuclear cells with banded, unsegmented nuclei from the old polymorphonuclear cells with segmented nuclei. An increased number of young cells indicates a response on the part of the bone marrow to an urgent demand for more polymorphonuclears. This is always present in a severe inflammatory condition. It has been our invariable experience that any marked increase in the young polys indicates acute inflammation, and if the inflammation continues, the old polys may be destroyed faster than the new ones can be delivered to the circulating blood. When this occurs the total leukocyte count may actually come down, while the percentage of polys remains high and the percentage of young forms continues to increase. In our experience this has been a very valuable aid in deciding for or against operation.

❦

Harlan Shoemaker, M. D. (1014 Wilshire Medical Building, Los Angeles).—Doctor Larson has presented twelve cases of rupture of the gall-bladder in which little, if any, defense has been made in the way of previous barriers of inflammatory exudates to protect the general abdominal cavity from a deluge of bile. These cases are difficult to diagnose, because the acute state of gall-bladder disease, which as a rule becomes chronic shortly, passes over with symptoms which are chiefly diagnosed as dyspepsia, gas, neuralgia, and even myocarditis.

As the disease progresses over many months or even years, gall-stones are developed. These gall-stones are without symptoms, or may be accompanied by a mild dyspepsia. Some 20 per cent can be diagnosed without x-ray. The accuracy of diagnosis is much greater when the dye is given intravenously than by mouth. About 20 per cent of the stones are nonfunctioning, and can only be diagnosed at operation. This is the type of gall-bladder that gives some pain in the back, and is frequently mistaken for anginal pain.

At the terminal stage, an acute, purulent, gangrenous inflammation from a stone in the gall-bladder, an acute pancreatitis or perforation from malignancy occurs, generally in the latter decades of life, and most frequently is preceded by the effects of symptoms over a long period of time. Gall-bladder colic that continues throughout the day is most frequently associated with an impacted stone. On the other hand, with the mild general symptoms that accompany a rupture of the gall-bladder in the earlier stages of gall-bladder disease, the average surgeon will advise the patient to wait until there is a sharper demarcation in his disease.

If this gall-bladder has ruptured, as Doctor Larson suggested, when the more advanced symptoms begin to appear, very little if any aid can be had by surgery or any other means at our disposal. Patients who are operated on within five days following an attack have generally recovered. Subsequent to that time, the disease is invariably fatal.

If the patients become seriously jaundiced, about 20 per cent will suffer from secondary hemorrhages. There is nothing that I know of that will ameliorate this condition except a blood transfusion.

In skilled hands, cholecystectomies should not have more than a one per cent mortality. Therefore, it is difficult to understand why the profession at large should not advise an exploratory procedure rather than to wait for a surgical impasse.

❦

Stanley H. Mentzer, M. D. (450 Sutter Street, San Francisco).—Acute perforations of the gall-bladder, which occur as exacerbations of chronic cholecystic disease, characterized by a long history of biliary disturbance, rarely become diagnostic problems. But the sudden acute perforations which occur in a previously "silent" gall-bladder cause serious diagnostic errors. Equally misleading are those acute perforations which give little clinical evidence of their occurrence. Unfortunately, these are more common than are generally supposed. In the twelve cases recorded by Doctor Larson, two belong to the former group and one to the latter, with the result that a cholecystic lesion was preoperatively diagnosed in only two instances. I have previously reported that four of twenty-four perforations were not even operated upon, and six were not preoperatively considered perforations. More recently perforation was diagnosed in only three of fourteen instances. Doctor Larson's experience, and my own, seem to indicate that our conception of the clinical signs of gall-bladder perforation is erroneous. And indeed, ample evidence has accumulated to prove that acute cholecystitis may be present without the history, physical examination and laboratory aids being characteristic of acute abdominal disease.

To avoid the high mortality of 50 per cent reported by Doctor Larson and others, we must familiarize ourselves with the eccentricities of the clinical evidence in acute and perforative cholecystitis. Until that is accomplished, earlier operative exploration in every doubtful case is warranted.

❦

Doctor Larson (closing).—I wish to thank the discussants for their excellent comments.

I will state again that this paper emphasizes the gall-bladder perforation that leads to generalized choleperitoneum; that the diagnosis is infrequent; that the surgical attack is too often not well planned, and that the mortality is much too high.

Doctor Chandler's suggestion that the Schilling blood test may be an important adjunct in establising a proper diagnosis should not be overlooked.

The discussions by Doctor Shoemaker and Doctor Mentzer should be carefully studied, since they both have had much experience with this condition.

HEART DISEASE COMPLICATING PREGNANCY*

By Clarence A. DePuy, M.D.
Oakland

Discussion *by James V. Campbell, M.D., Oakland; Bernard J. Hanley, M.D., Los Angeles; E. A. Royston, M.D., Los Angeles.*

THE matter of heart disease as a complication of pregnancy is always one to give the obstetrician cause for serious thought. Is the patient's life in grave danger; is it necessary to interrupt pregnancy, or is it possible to decide whether there are circumstances under which the expectant mother may, without great risk, be permitted to go on to term?

* Read before the Obstetrics and Gynecology Section of the California Medical Association at the sixty-fourth annual session, Yosemite National Park, May 13–16, 1935.